The differences indicate that A/Vietnam/14011801/2014 is a novel reassortant virus between clades 2.3.2.1a and 2.3.2.1c, between clades 1.1.2 and 2.3.2.1c, or both (Figure). This novel reassortant virus has not been reported in poultry in Vietnam, although novel reassortants between clade 1.1.2 and clade 2.3.2.1a viruses have been detected in Vietnam since 2013 (i.e., A/Vietnam/VP13-28H/2013, GISAID accession nos. EPI624927–EPI624934; and A/Vietnam/14012902/2014) (*6*). These novel reassortment viruses were first identified in human, animal, and environmental samples in Cambodia in 2013 (*5*). Other novel gene reassortments in clade 2.3.2.1 viruses have been previously reported (*10*), and new clade 2.3.4.4 viruses have been observed in Vietnam since 2014.

As multiple clade viruses co-circulate, reassortment events occur frequently in Vietnam. Continuous surveillance of avian influenza A(H5N1) viruses, not only in humans but also in poultry and wild birds, is needed for infection control measures during epidemics of these viruses.

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Mycobacterium arupense as an Emerging Cause of Tenosynovitis

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To the Editor: *Mycobacterium arupense* was identified in 2006 as a novel species within the *M. terrae* complex with close similarity to *M. nonchromogenicum* (*1*). Since then, 8 cases describing clinically notable disease have been published (*2–8*), including 5 cases of tenosynovitis. We report *M. arupense* tenosynovitis in an immunocompromised person who received the selective interleukin (IL) 1 β-inhibitor canakinumab.

In July 2014, a 62-year-old man sought treatment at the emergency department, Northwestern Memorial Hospital (Chicago, Illinois, USA), after 1 week of pain and swelling in the right hand. During the previous 5 years, he had received multiple immunomodulatory drugs for treatment of natural killer cell deficiency, hyper–IL-6 syndrome, recurrent polychondritis, and Sweet syndrome. His medications were prednisone (42.5 mg/d), intravenous immunoglobulin (400 mg/kg monthly), and subcutaneous canakinumab (180 mg every 8 weeks, which began 3 weeks before onset of symptoms).

His first symptom was a tender red nodule on the right palm that increased in size and became extremely tender over the following week (Figure, panels A, B). He did not recall any trauma and denied fever or chills. No improvement was seen after he received oral linezolid for 5 days. A

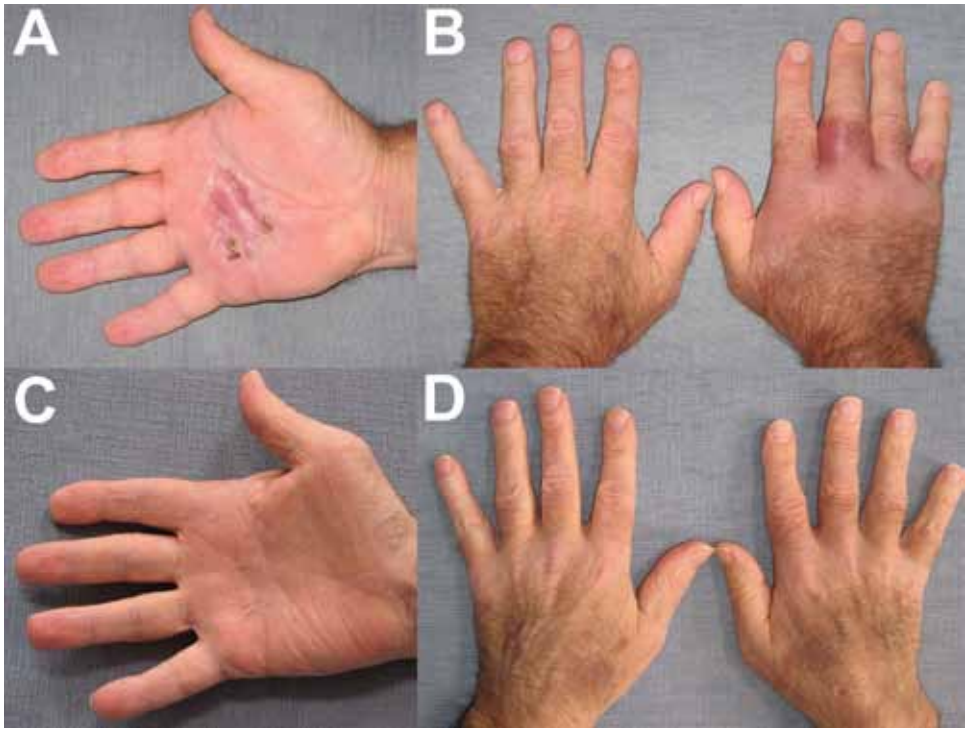

Figure. Hands of a 62-year-old man in Chicago, Illinois, USA, who had *Mycobacterium arupense* tenosynovitis, at the time treatment was sought (panels A, B) and after 6 months of treatment (panels C, D).

skin punch biopsy specimen showed a neutrophilic interstitial infiltrate with no granulomas; results of microbiological stains, including acid-fast bacilli, were negative, . His prednisone dosage was increased to 60 mg/d for suspected Sweet syndrome and, subsequently, to 80 mg/d when no improvement was observed after 2 weeks. A second dose of canakinumab was administered 8 weeks after the first. Shortly after, he was readmitted to the hospital with progression of edema and pain and signs consistent with carpal tunnel syndrome and trigger finger syndrome of the right index finger. Magnetic resonance imaging showed extensive tenosynovitis of the carpal tunnel flexor tendons and no bone erosions. Surgical release and tenosynovectomy of the carpal tunnel was performed; pathologic features demonstrated chronic inflammation of the synovium and absence of granulomas. Results of microbiological stains were negative.

M. arupense grew on Löwenstein-Jensen culture from the skin biopsy specimen after 35 days and from a synovium specimen after 22 days. No growth was observed on liquid culture media. Empiric treatment was started immediately after the first positive culture: clarithromycin (500 mg 2×/d), ethambutol (1,200 mg/d), and rifabutin (300 mg/d). Prednisone was decreased to 45 mg/d, and canakinumab was discontinued. Susceptibility testing confirmed the *M. arupense* strain's susceptibility to clarithromycin, ethambutol, and rifabutin (MICs <4.0, <1.25, and <0.12, respectively); intermediate resistance to rifampin and amikacin (MIC 4.0); and resistance to moxifloxacin and ciprofloxacin (MIC >4.0) and to kanamycin (MIC >8.0). Clinical improvement occurred after 8 weeks of treatment; the condition resolved after 6 months (Figure, panels C, D). Treatment was continued for 12 months.

Five other cases of *M. arupense* tenosynovitis have been reported (*2*,*4*,*5*,*7*,*8*); all patients were immunocompetent or minimally immunocompromised (i.e., diabetes mellitus) (online Technical Appendix, http://wwwnc.cdc.gov/EID/article/22/3/15-1749-Techapp1.pdf). The hand was the site of infection in all cases, and 4 of 5 patients reported prior trauma to the affected area, which suggests that inoculation was the infection mechanism. In the case we describe, the disease appeared to progress much faster than in the immunocompetent patients (weeks vs. months to years). Acid-fast bacilli stain was negative in all of the cases where it was performed (*2,7,8*; this study), and growth on solid Löwenstein-Jensen stain or Middlebrook media was seen after a prolonged incubation time, ranging from 27 days to 2 months. Liquid culture media appears to be unreliable for the growth of *M. arupense* (*8*; this study).

A combination of tenosynovectomy and prolonged antimycobacterial treatment, guided by in vitro strain susceptibility, was used in all the reported cases; a positive outcome was achieved in 6–14 months. The strain susceptibility results we found are comparable with those in the previous cases, showing consistent susceptibility to clarithromycin, ethambutol, and rifabutin; variable susceptibility to linezolid, streptomycin, and amikacin; and resistance to rifampin and quinolones.

Two cases of *M. arupense* infection have been reported in immunosuppressed persons, both in HIV/AIDS patients (manifesting as pulmonary infection in 1 patient and disseminated disease in the other) (*6*). In our study, the immunocompromised patient with *M. arupense* tenosynovitis received canakinumab, a relatively new biologic agent with a prolonged selective IL-1 β-blockade. Even though the contribution of canakinumab in this case is confounded by concomitant immune deficiencies (natural killer cell deficiency, high-dose corticosteroids), the temporal association between initiation of canakinumab and the onset of symptoms raises concern of a possible association. Animal studies have shown that IL-1 plays a key role in host resistance to mycobacterial infections by regulating Th1/Th2 immune responses and inducing granuloma formation (*9*). Clinical trials and systematic reviews assessing the safety of IL-1 inhibitors, including anakinra, rilonacept, and canakinumab, have not shown that these drugs lead to an increased risk of tuberculosis or other mycobacterial infections (*10*). Nonetheless, our report provides increased evidence that *M. arupense* is an emerging cause of tenosynovitis and that it is potentially associated with immunosuppression.

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Candida haemulonii Complex Species, Brazil, January 2010–March 2015

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To the Editor: The epidemiology of yeast infections is evolving, and species in the *Candida haemulonii* complex have been identified as a cause of candidiasis (*1*). In 2012, *C. haemulonii* complex was reclassified as 2 species and 1 variety: *C. haemulonii* (former group I), *C. duobushaemulonii* (former group II) and *C. haemulonii* var. *vulnera* (*1*).

Despite the growing knowledge about the biology and clinical relevance of these pathogens, species-specific data comparing clinical and microbiological aspects are lacking. We describe the clinical and microbiological characteristics of patients from 5 hospitals in São Paulo, Brazil, whose cultures were positive for the *C. haemulonii* complex species.

During January 2010–March 2015, samples from case-patients in 5 hospitals affiliated with the University of São Paulo were cultured; samples positive for *C. haemulonii* were further analyzed. Clinical and epidemiologic data were retrospectively collected. Species identification of the first isolate from each patient was made by sequencing the internal transcribed spacer region of the rRNA gene (*2*). Sequence similarity searches were done by using BLAST (http://www.ncbi.nlm.nih.gov/blast). Antifungal susceptibility testing was performed by using the Clinical

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Mycobacterium arupense as an Emerging Cause of Tenosynovitis

Technical Appendix

Technical Appendix Table 1. Patient clinical characteristics of case-patients with *Mycobacterium arupense* tenosynovitis* in published reports

Report	Year	Country	Sex, age, y	Coexisting condition	Presentation	Initial event	Risk for progression	Symptom duration, wk†
Tsai et al. (*1*)	2008	Taiwan	F, 54	Diabetes mellitus	TS/hand	Blunt trauma		28
Senda et al. (*2*)	2011	Japan	M, 68	Hypertension	TS/hand	None	Corticosteroid injection	20
Legout et al. (*3*)	2012	France	M, 35	None	TS and OM/ wrist	Penetrating trauma (glass)	Corticosteroid injection	68
Lee et al. (*4*)	2014	North Korea	F, 56	Hypertension, resected pituitary adenoma. Receiving low-dose prednisolone.	TS/hand	Penetrating trauma (by crab)	Corticosteroid injection	44
Beam et al. (*5*)	2014	USA	M, 58	None	TS/hand	Remote blunt trauma	Systemic corticosteroids, methotraxate, adalimubab	100
This report	2015	USA	M, 62	NK cell deficiency, hyper IL-6 syndrome, recurrent polychondritis, Sweet syndrome. Receiving high- dose prednisone and canakinumab.	TS/hand and wrist	None	Increasing dose of systemic corticosteroids	6

*TS, tenosynovitis; OM, osteomyelitis; NK, natural killer; IL, interleukin.
†Time reported from initial symptoms to diagnosis.

Technical Appendix Table 2. Microbiological and treatment characteristics of case-patients with *Mycobacterium arupense* tenosynovitis in published reports *

Case	Pathology	Culture	Identification method	Susceptibility	Resistance	Treatment	Treatment duration, mo	Outcome
Tsai et al. (*1*)	Granulomas+, AFB–	L-J positive at 60 d	16S rRNA, hsp65 and rpoB	NR	NR	Synovectomy. clarithromycin, ethambutol, rifabutin, moxifloxacin, ciprofloxacin	6	Resolved
Senda et al. (*2*)	Granulomas+, AFB NR	L-J negative	DNA-DNA hybridization	NA	NA	Synovectomy. ethambutol, rifampin	14	Resolved
Legout et al. (*3*)	Granulomas+, AFB NR	L-J positive	16S rRNA and hsp65	NR	NR	Synovectomy and arthrodesis. Clarithromycin, ciprofloxacin, amikacin (1 mo), ethambutol (2 mo)	12	Resolved
Lee et al. (*4*)	Granulomas+, AFB–	L-J positive at 27 d	16S rRNA and hsp65	Clarithromycin, ethambutol, linezolid	Ciprofloxacin, moxifloxacin, rifampin,	Synovectomy. clarithromycin, ethambutol, rifampin	NR	Resolved

Case	Pathology	Culture	Identification method	Susceptibility	Resistance	Treatment	Treatment duration, mo	Outcome
Beam et al. (5)	Granulomas–,AFB–	MGIT negative L-J positive at 33 d	16S rRNA	Clarithromycin, ethambutol, rifabutin	amikacin, TMP/SMX Ciprofloxacin, moxifloxacin, rifampin, amikacin, TMP/SMX, linezolid	Synovectomy. clarithromycin, ethambutol, rifabutin	6, ongoing	Improved
This study	Granulomas–,AFB–	MGIT negative L-J positive at 35 d	16S rRNA			Synovectomy. clarithromycin, ethambutol, rifabutin	12	Resolved

*AFB, acid-fast bacilli staining; L-J, Löwenstein-Jensen culture; MGIT, mycobacteria growth indicator tube; NR, not reported; NA, not applicable; TMP/SMX, trimethoprim/sulfamethoxazole.
†Time reported from initial symptoms to diagnosis.

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